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Current and Future Management Strategies of Type A Aortic Dissection

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Abstract

Type A Aortic dissection is a life-threatening emergency. It has varied clinical presentation from acute severe chest pain radiating to the back, collapse due to aortic rupture or pericardial tamponade or features of myocardial infarction, end organ or limb ischemia. The outcome is determined by the extent of the dissection, timing of presentation, comorbid factors, prompt diagnosis, adequate cerebral protection strategies, and skilled post-operative intensive care. Good immediate and mid-term results have been obtained with standard surgical techniques of aortic root, ascending aorta +/- hemi arch replacement. Endovascular techniques can be used as a hybrid procedure to provide more durable long term results.

Keywords: dissection, cerebral protection strategies, aortic root, ascending aorta, hemi arch, endovascular techniques

1. Introduction

Type A aortic dissection is a life threatening condition requiring emergency surgical intervention. Statistics show an incidence of approximately three cases per 100,000 per year [1, 2]. Data analysis from the International Registry of Acute Aortic Dissections (IRAD) reported a predominant male patient population and a mean age of 63 years at presentation [3]. Surgical repair for Type A Aortic dissection is challenging, the complexity proportionate to the location and extent of aortic tissue dissected, cardiac complications and end organ ischemia. The current standard surgical approach includes replacement of the aortic root (Bentall technique) or valve sparing root replacement, isolated ascending aorta replacement, and hemi or full arch replacement. Recent advances include frozen elephant trunk (FET) technique, total aortic repair, endovascular and hybrid approaches and stenting. All of these surgical approaches, including classification, clinical presentation, risk factors, diagnosis, pre-operative preparation, cannulation strategies, and cerebral protection will be discussed in this review.

2. Classification

The Stanford classification (1970) is the most commonly used system [4] (Figure 1). It does not classify the site of tear. It is more of a clinically useful

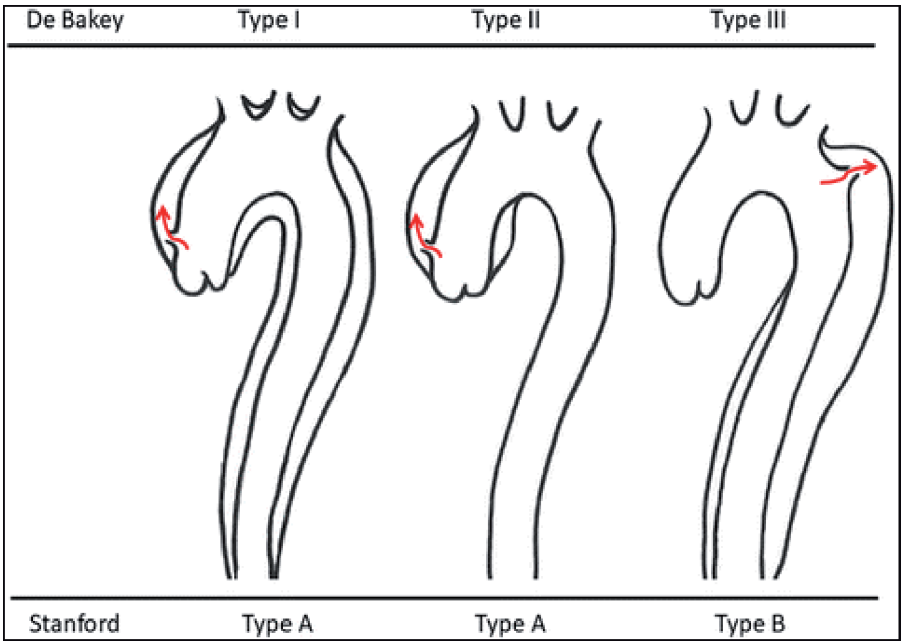


Figure 1.
Stanford and DeBakey classification.

classification to guide acute management. Stanford type A involves ascending aorta proximal to innominate artery, irrespective of involvement of aortic arch or descending aorta. Stanford type B dissection involves only the thoracic aorta distal to the left subclavian artery [5]. Intimal tears originating distal to left subclavian artery but dissecting retrogradely into ascending aorta will be type A. Intimal tears starting in the aortic arch and extending into ascending aorta are type A and extending into descending aorta are type B. If the intimal tear is restricted to aortic arch, then it is type non-A non-B.

The DeBakey type I involves ascending aorta and extends beyond the innominate artery. Type II involves only the ascending aorta. Type III involves thoracic aorta distal to left subclavian artery. It is more useful for long-term follow-up as it differentiates between proximal and distal aortic dissection extent (**Figure 1**) [4].

The Penn classification is a recently introduced method of classifying based on clinical presentation [6]. Class Aa—absence of branch vessel malperfusion or circulatory collapse; Class Ab—symptoms or signs of localised organ ischemia; Class Ac—circulatory collapse with or without cardiac involvement; Class Abc—combined localised and generalised ischemia.

3. Risk factors

Men form approximately 62–68% of all patients undergoing surgery [7]. The triad of hypertension (67–86% patients), smoking and atherosclerosis is an independent predictor of type A aortic dissection [8]. Data correlating the risk of aortic dissection in bicuspid aortic valve is limited and controversial. However, some centres advocate early prophylactic ascending aortic replacement in patients with aortas larger than approximately 5.0 cm in diameter or with a cross-sectional area to height ratio greater than approximately 10 cm²/m [9]. In the NORCAAD registry, 6% patients were reported to have bicuspid aortic valve [7]. The Marfan syndrome is present in around 4% of ATAAD patients in NORCAAD registry [10]. The patients typically have pear-shaped aneurysm of aortic root. Due to mutation in FBN1 gene, there is decreased strength and elasticity of elastin—rich tissue of aortic wall. Predominantly, medium and large sized arteries are involved in dissection (**Table 1**).

(1) Cardiovascular and life style factors
Uncontrolled hypertension
Age
Smoking
Dyslipidemia
Cocaine
Pregnancy
(2) Congenital and connective tissue
Bicuspid aortic valve
Marfan syndrome
(3) Iatrogenic
Coronary catheterisation
Arterial cannulation
Aortic cross clamping
IABP
(4) Vascular inflammation
Takayasu arteritis
(5) Trauma
Deceleration injury

Table 1.
Risk factors for acute aortic dissection.

4. Clinical presentation

TAAD patients (85%) typically present with sudden intense central chest pain (ripping or tearing) radiating to the neck, back or abdomen. [11]. ECG abnormalities (50% patients) include ST/T wave changes, conduction defects, and arrhythmias. This sometimes leads to the misdiagnosis of myocardial infarction leading to fatal mistake of giving antiplatelet therapy or thrombolysis. This delays the diagnosis and increases peri-operative bleeding complications. Aortic regurgitation (70% patients) can occur through prolapse of right or non- coronary valve cusps and detachment of commissures. Pulmonary oedema may occur through acute dilation of the left ventricle. Haemorrhagic pericardial tamponade is a very strong predictor of aortic dissection. Reduced or absent femoral pulses are seen. Neurological sequelae include syncope, stroke. Mal-perfusion of spinal cord leads to paraplegia. Acute renal failure and mesenteric ischemia are seen if the dissection involves descending thoracic and abdominal aorta [12]. Rupture of the aneurysm is immediately fatal.

5. Diagnosis

1. **Clinical:** Constellation of findings of chest pain, diastolic murmur, blood pressure difference in both upper limbs, pulse deficit, neurological sequelae are good indicators for TAAD [13].
2. **Chest X ray (Figure 2)** shows widened mediastinum (49% patients), which is non-specific [14].

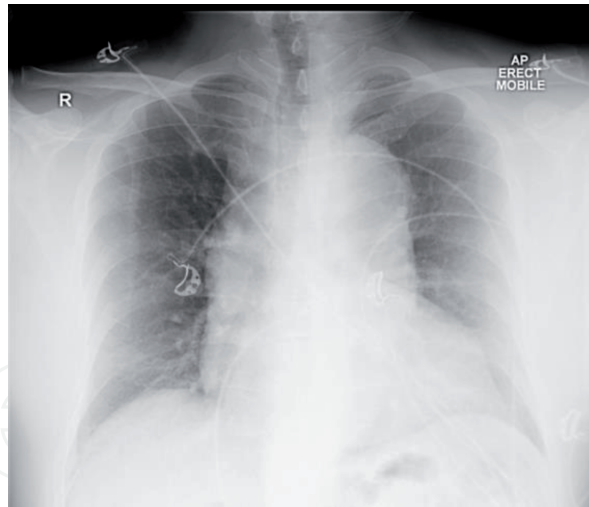


Figure 2.
Chest X-ray of a patient with ATAD showing a widened mediastinum.

3. **ECG** may show ST-T changes especially in right coronary territory, non-specific changes of left ventricle hypertrophy, pericardial effusion
4. **CT scan (Figure 3):** the most reliable diagnostic tool for acute aortic dissection. It is available in almost every hospital and can be rapidly performed. Sensitivity and specificity is excellent [15]

The following information may be provided from a CT-scan:

- a. Detection of the true and false lumen in the dissected aorta
- b. Identification of the site of the intimal tear The extent of the dissection
- c. Arch vessel and thoracic and abdominal branch vessel involvement
- d. Planning the site of cannulation

Limitations of CT-scanning include not providing information about dissection entry site and functional status of the heart.

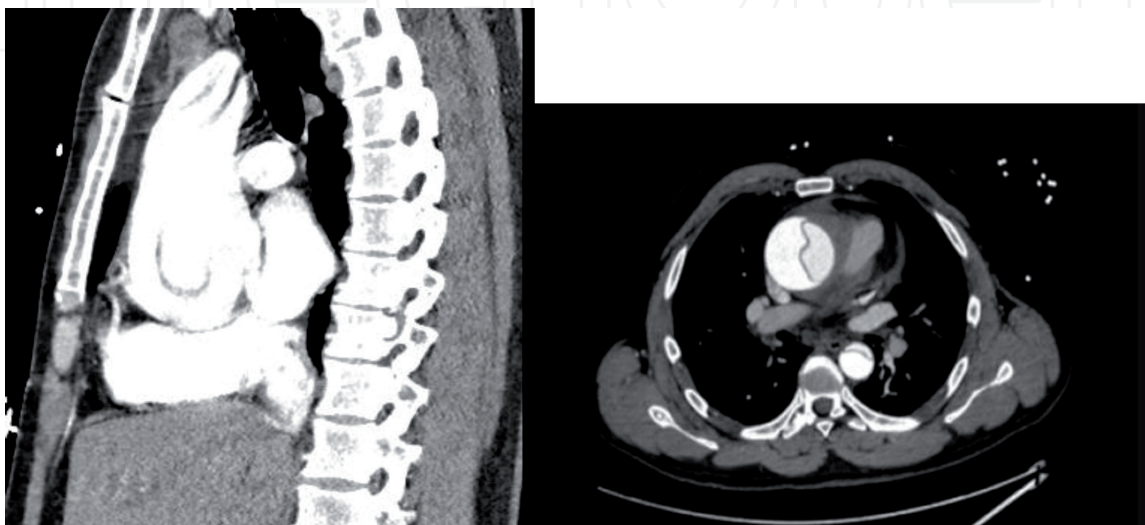


Figure 3.
CT-scan of a patient with TAAD.

5. Trans-oesophageal echocardiography (TEE; **Figure 4**)

TEE is portable, less invasive and has sensitivity and specificity approximately 100% [16]. It provides information about:

- a. The dissection flap and false lumen in ascending aorta
- b. The entry site of tear using colour Doppler
- c. Coronary ostial obstruction due to the dissection
- d. Dilation of aorta and left ventricle function
- e. Pericardial effusion and tamponade
- f. Aortic regurgitation and anatomy of aortic root
- g. Has a better window than TTE to visualise aortic arch and descending aorta
- h. Pleural effusion

Limitations include difficulty in visualisation of the proximal arch due to the interposition of bronchial air [17].

6. Magnetic resonance imaging (MRI)

MRI is an accurate investigative modality for acute aortic dissection (sensitivity and specificity, 98%) [18]. It is rarely used in the setting of TAAD where most of the patients are wheeled into operating room as soon as the diagnosis is made. It may have a small role in those patients allergic to iodinated contrast agents or in patients with acute renal failure who are stable enough to undergo MRI.

7. Preoperative coronary angiography:

Its role is controversial [19]. Justification of not performing cardiac catheterisation include risk of catheter induced aortic rupture and delaying surgery where percentage mortality increases by 1 % every hour [20].

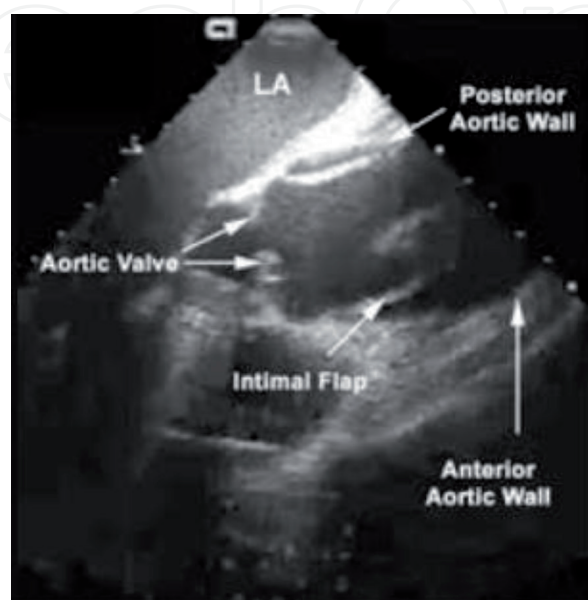


Figure 4.
TEE of a patient with ATAD.

6. Surgical strategies

6.1 Preparation for surgery

1. Blood pressure (BP) control: One of the most important pre-requisites of successful outcome is very strict control of BP. Systolic BP should be less than 110 mmHg. It can be lowered using intravenous beta blocker (esmolol) or combined alpha & beta receptor antagonists (labetalol) or glyceryl trinitrate. Intravenous adrenaline should be used in patients presenting with cardiogenic shock and cardiac tamponade. Anaesthetic induction is also another step where BP should be tightly controlled. Commonly used medications include Fentanyl, isoflurane and glyceryl trinitrate. Peri-operatively, a target systolic blood pressure of 90–110 mmHg, mean arterial pressure of 60 mmHg, and central venous pressure of 8–12 mmHg are recommended [21].
2. Coagulation status: Aortic dissection activates inflammatory, coagulation and fibrinolytic pathways leading to disseminated intravascular coagulation. Consumption coagulopathy is worsened in some patients by inadvertently prescribing aspirin, clopidogrel by misdiagnosing these patients as having acute coronary syndromes. Hence, adequate amounts of packed red blood cells, platelets, fresh frozen plasma and cryoprecipitate should be kept ready for use.
3. 2 arterial lines (both arms) should be placed to monitor differential blood pressures.

Also include a femoral arterial line to monitor distal perfusion pressures
4. Cerebral oxygenation monitoring by near—infrared spectroscopy
5. Invasive monitoring of intracranial pressure by lumbar catheter (occasionally)

7. Cannulation strategies

1. Femoral artery cannulation (**Figure 5**)

For many years, it has been the cannulation site of choice [22]. Allowing rapid access, it is usually used in hemodynamically unstable patients, especially with impending cardiac tamponade and aortic rupture. It is important to mark the femoral artery before skin preparation as it may be difficult to localise it during hypotension. Common femoral artery is situated medial and inferior to the midpoint of the inguinal ligament. An oblique or vertical incision may be used for exposure [23]. An open Seldinger technique is quick and can be performed with minimal dissection. Advantages of this approach include (i) cardiopulmonary bypass is established quickly, (ii) easy to access with a closed chest, (iii) less likely to be dissected, (iv) prevents aortic rupture in patients with cardiac tamponade. Disadvantages include (i) stroke and malperfusion due to dynamic obstruction and (ii) retrograde perfusion leading to embolic complications due to atherosclerotic emboli. The femoral artery with a dissection flap is not used for cannulation.

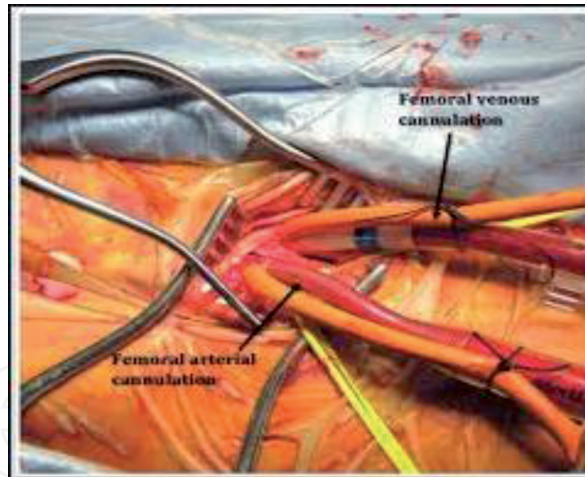


Figure 5.
Femoral artery cannulation.

2. Axillary artery cannulation (**Figure 6**)

Introduced in 1990s [24], axillary artery cannulation is gaining greater acceptance among surgeons as they switch to an antegrade perfusion strategy. It is more commonly used in hemodynamically stable patients. Infraclavicular dissection exposes the first part of axillary artery [25]. The pectoralis major muscle is split. The neurovascular bundle is situated deep in the clavipectoral fascia. The deltopectoral approach exposes the second and third parts for cannulation [26]. The axillary artery can be directly cannulated or anastomosed with end to side 8 mm vascular graft. Advantages include (i) antegrade perfusion flow and (ii) can be used for antegrade cerebral perfusion by occluding innominate artery. Disadvantages of this approach include (i) takes more time than femoral cannulation especially in obese patients and (ii) technically more difficult and risk of injury to brachial plexus nerves.

3. Central aortic cannulation:

Locating a site where the chances of not entering into the false lumen is the most critical part. It can be done with TEE, CT or epiaortic scanning. Cannulation can be performed with Seldinger technique or directly. Advantages include (i) CPB

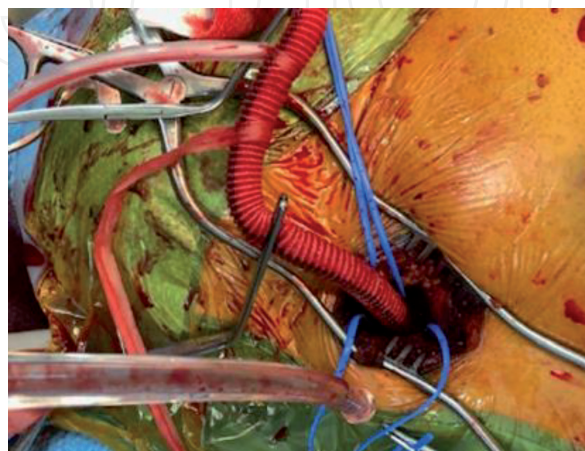


Figure 6.
Axillary artery cannulation.

established quickly in unstable patients and (ii) antegrade flow. Disadvantages include (i) rupture of cannulation site and (ii) false lumen perfusion [27].

8. Peri-aortic dissection before Going on CPB and after cardiac arrest

1. An expeditious midline sternotomy should be done with SBP maintained around 100–110 mmHg. The dissected aorta is usually dilated, thinned out and blood seeping through adventitial layers (**Figure 7**).
2. Upon opening the pericardium, be prepared for free aortic rupture (**Figure 8**). To minimise the risk either (i) femoral arterial and venous cannula should be in place or (ii) if axillary artery cannulation is done, then the surgeon should be prepared to quickly place the two-stage cannula into right atrium.
3. Patient is cooled to a core temperature of 26–28°C. For hemi-arch replacement and short duration of total hypothermic circulatory arrest, 27°C temperature is optimum. If total arch replacement is planned, then the patient can be further cooled to 22–24°C.
4. After going on CPB, a left ventricular vent is placed to prevent left ventricular distension due to associated acute aortic regurgitation. Retrograde cardioplegia catheter is placed to arrest the heart, as antegrade cardioplegic arrest may not be possible due to aortic regurgitation. After the cardiac arrest and upon opening the aorta, antegrade ostial cardioplegia can be administered.
5. If the patient is stable, innominate artery is carefully dissected and looped with a vascular loop before going on CPB. This can be occluded or clamped later to provide antegrade cerebral perfusion through the previously cannulated axillary artery.
6. After going on CPB, the aorta is dissected free from surrounding adhesions. When creating the plane between ascending aorta and main pulmonary artery, it is important to retain as much adventitial tissue on the aortic side.
7. It is important to identify right pulmonary artery and avoid injuring it, while dissecting ascending aorta.

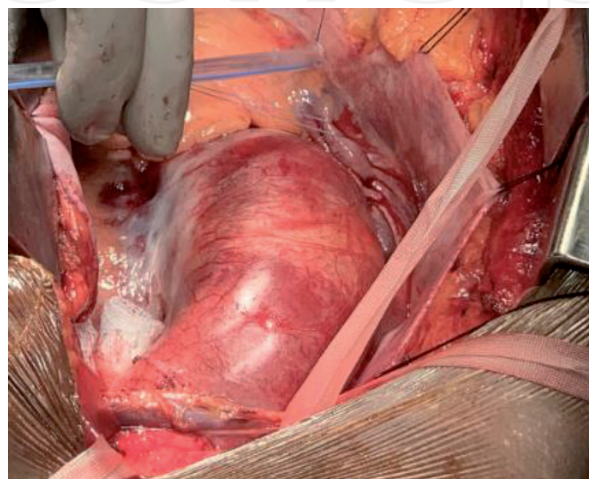


Figure 7.
The dissected ascending aorta.

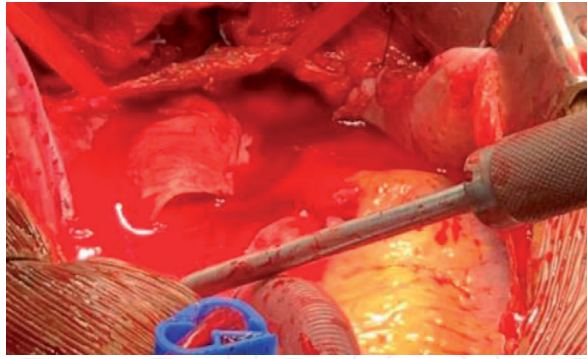


Figure 8.
Aortic rupture prior to establishing CPB.

8. Clamp the aorta somewhere in the mid-ascending aorta, which will be eventually resected while doing open distal anastomoses. This allows assessment of the site of the tear and of the aortic root and also minimises the total circulatory arrest time.
9. While excising the dissected ascending aorta, it is important to avoid injury to the main and right pulmonary artery.
10. While dissecting towards aortic root, left and right coronary ostia are identified and coronary buttons prepared (if aortic root replacement is planned).

9. Hypothermic circulatory cardiac arrest and cerebral protection strategies

Because of the low threshold tolerance to ischemia, brain protection is of paramount importance during aortic arch procedures. Hypothermia is an option to increase the ischemic time. However, there are limitations of hypothermia and hypothermic circulatory arrest. Protective effects of hypothermia last no more than 9 minutes at 30°, 14 minutes at 25°, 21 minutes at 2°, 31 minutes at 15° and 45 minutes at 10° [28]. Neurological deficits are seen in elderly patients subjected to hypothermic circulatory arrest exceeding 25 minutes.

Several cerebral perfusion techniques have been introduced to extend the safe period of arch repair without residual neurological deficits. Retrograde cerebral perfusion (RCP) in tandem with hypothermic circulatory arrest was introduced in 1990 by Ueda et al. [29] Because the cerebral venous sinuses have no valves, RCP was proposed to provide retrograde perfusion and cooling of central nervous system (CNS). It offered to back-flush air emboli and debris from the cerebral circulation. Neuroprotective effects were most likely related to cooling rather than true nutritive flow [30]. It was also found to provide limited benefit in patients with significant carotid stenosis and vascular anomalies (e.g. an incomplete Circle of Willis) [31].

Antegrade selective cerebral perfusion (SCP) was introduced by Jean Bachet and Daniel Guilmet in Europe [32] and by Teruhisa Kazui in Japan in 1986 [33]. This new perfusion method of “cold cerebroplegia” in combination with hypothermia significantly reduced neurologic complications. Antegrade selective perfusion can be established either by direct cannulation or by anastomosing a prosthetic graft. The options for locating such are (i) right subclavian artery, (ii) innominate artery, (iii) right common carotid artery. These may be combined with left common carotid artery cannulation to provide bilateral antegrade cerebral perfusion. Direct

cannulation is limited by high risk of embolism due to plaque mobilisation from manipulation or by jet flow [34].

Axillary artery cannulation can be used to provide unilateral antegrade SCP during hypothermic arrest without manipulation of the arch vessels. This can be combined with balloon occludable perfusion catheter to left carotid artery to provide bilateral antegrade SCP. To avoid steal, an occlusive balloon catheter is inserted in left subclavian artery.

Unilateral antegrade SCP is sufficient for majority of patients with no pathology of the arch vessels and cerebral vessels. Adequate backflow from the contralateral carotid artery suggests good collateralisation. Near-infrared spectroscopy (NIRS) monitoring can also help to exclude contralateral malperfusion. Bilateral cerebral perfusion may be useful in patients with carotid artery stenosis, previous stroke or cerebrovascular anomalies (incomplete Circle of Willis). Malvindi concluded in his review that “While both unilateral and bilateral ASCP are acceptable, bilateral antegrade cerebral perfusion is safer, when the antegrade SCP time is more than 40-50 minute” [35].

Cerebral perfusion is performed at a rate of 8–12 cc/min/kg body weight, perfusion pressure of 40–60 mmHg at 23–28°C. Alpha stat pH management compared to pH stat management prevents “luxury perfusion” by maintaining cerebral autoregulation decreasing the risk of embolization [35].

10. Technical aspects of surgical repair

10.1 Aortic root management

The aortic root is frequently involved with aortic valve rendered incompetent due to commissural dehiscence or annular dilation. Grade II and grade III aortic regurgitation was found in 40 and 23% patients, respectively, in German Registry for Acute Aortic Dissection type A (GERAADA) [36]. There are different surgical approaches for management of aortic root replacement—an aggressive or a more conservative approach.

According to the International Registry of Acute Dissection (IRAD), aortic root replacement compared with conservative root management is not associated with increased in-hospital mortality. In 1995 patients, 18 and 21.3% hospital mortality was found in root replacement and conservative root group respectively (OR 0.989; CI 0.710–1.379; $p = 0.949$). Mid-term results at 3 years showed a survival of 91.6±1.3% and 92.5±1.7% for conservative root management and root replacement group, respectively [37].

Indications to perform root replacement in a patient with ATAD include:

1. younger age
2. Marfan syndrome
3. bicuspid aortic valve
4. known aortic valve disease
5. moderate or severe aortic valve insufficiency
6. previous aortic valve replacement
7. large diameter aortic annulus, sinuses of Valsalva and ascending aorta

8. coronary artery involvement

9. aortic root as the most proximal site of dissection

Proximal reconstruction technical (**Figures 9** and **10**) details: After the excision of the dissected aortic root, aortic root reconstruction is done.

Technical principles include:

- i. Obliteration of false lumen.
- ii. To take the maximum possible adventitia in the anastomoses.
- iii. Needle should enter the tissue at right angle to avoid tearing of needle holes.
- iv. Sutures should be spaced uniformly and pulled just enough tight to prevent cutting through the fragile tissue.
- v. To avoid distortion of the aortic valve, the planar relationship between the graft and sinotubular junction should be maintained.

The Bentall procedure [38] is a fairly standard procedure. It includes anastomosing the composite graft to the aortic annulus and reimplantation of coronary arteries. Patients with type A aortic dissection usually have normal aortic valve cusps, as the pathology is usually limited to the aortic wall.

Valve sparing root replacement is a viable option in hemodynamically stable young patients. But it is technically more complex than straight aortic root replacement. It involves replacing the aortic root by composite graft without replacing the aortic valve. The native aortic root must be dissected from surrounding structures to 2 mm below the nadir of the aortic annulus. Coronary ostia are reimplanted into the graft. It is used to treat aortic regurgitation due to annular enlargement. Contraindications include significant cardiomyopathy, malperfusion, coronary artery disease, >65 years age.

Results from Emory in a 43 patients showed operative mortality of 4.7%. Freedom from aortic valve replacement was 100% and freedom from more than mild aortic regurgitation was 94% at 9 years follow-up. No aortic root reinterventions were required in this series [39].

Conservative approaches to the aortic root (CRR)—In most of the patients presenting with TAAD, the most common pathology seen is a primary intimal tear in the ascending aorta with dissection flap extending to non-coronary cusp. Left and right coronary sinuses are relatively preserved. Any aortic regurgitation



Figure 9.
Aortic root replacement (right coronary ostial anastomosis).

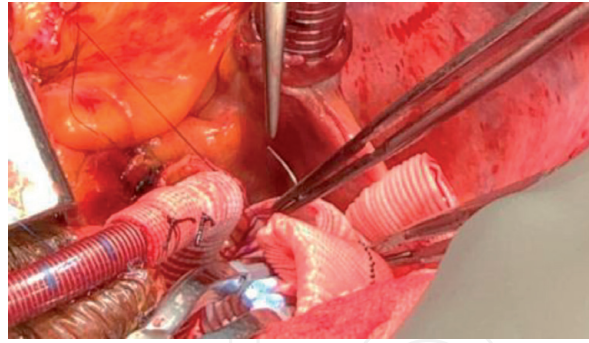


Figure 10.
Proximal anastomoses of aortic root.

is due to unhinging of one of the aortic valve commissural posts. The dissected sinus segments are preserved and supported with resuspension of the native valve commissural posts or prosthetic ascending aorta replacement. The advantages are that it preserves the native sinus tissue, coronary ostia are not reimplemented, shorter ischemic time, avoiding life-long anticoagulation [40]. The most commonly used methods to fortify the aortic wall include Teflon felt and biologic glue. University of Pennsylvania in their series of 489 patients showed freedom from reoperation with this technique of 96, 92 and 89% at 1, 10 and 15 years respectively. The operative mortality was 11% [41].

11. Ascending aorta

The entry site of the dissection tear is usually found the ascending aorta, which is at very high risk of rupture. After excising the dissected portion of ascending aorta, supracommissural ascending aorta replacement can be performed. Open distal anastomoses can be done under hypothermic circulatory arrest after releasing the cross clamp. This facilitates inspection of the aortic arch and if required, arch repair can be undertaken. Also, it is technically much easier to construct a very distal ascending aortic anastomoses. Around 5.6% patients underwent ascending aorta replacement in GERAADA survey [42]. But this procedure also allows for subsequent aneurysmal dilation of the remaining portion of the aorta [43].

12. Hemi-arch or full arch

When the aortic arch is examined during hypothermic circulatory arrest to look for intimal or re-entry tears, a decision is made whether an aortic arch replacement has to be done. The pre-operative CT aortogram helps in localising the dissection and also on deciding the placement of the aortic cross clamp. If the tear is in the ascending aorta or start of the aortic curvature, then hemi-arch replacement is required. If the dissection extends more distally, a total aortic arch repair should be performed (**Figure 11a, b**).

Indications for arch replacement include:

1. Pre-existing aneurysm of the arch
2. Primary intimal tear in distal arch or descending thoracic aorta
3. Secondary intimal tear in arch >10 mm

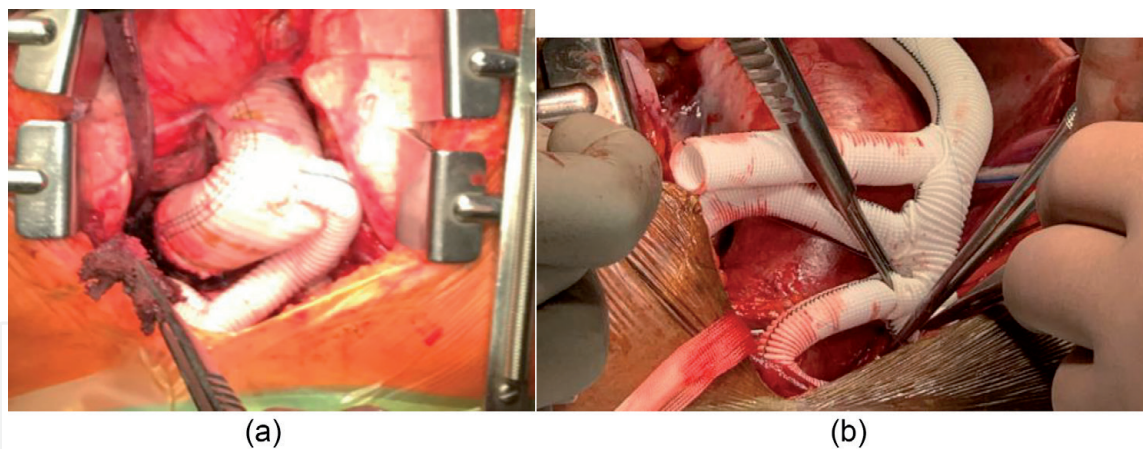


Figure 11.
 Total arch replacement. (a, b) Debranching of aortic arch vessels – innominate, carotid artery and left subclavian artery.

4. False lumen more than 22 mm

5. Descending thoracic aorta diameter more than 35 mm

A study of 188 patients by Kim et al. [44] showed that 5 year survival was lower in patients with total arch replacement compared to patients who had hemi-arch repair (65.8% vs. 83.2%, $p = 0.013$). Neurological complications were higher in total arch repair group compared to hemi-arch (56.9% vs. 24.8%, $p < 0.001$). There was a direct correlation between patent false lumen in aortic arch or descending aorta and re-intervention. The German registry for TAAD showed no significant difference in peri-operative outcomes between both groups [45]. This group suggested a more aggressive approach to reduce the rate of interventions.

13. Frozen elephant trunk (FET)

The immediate post-operative results have improved post type A Aortic dissection repair. However, long-term results are guarded by the need for aortic re-interventions due to residual dissection and patent false lumen extending into descending thoracic aorta [46]. The frozen elephant trunk technique involves total arch replacement and per-procedural deployment of stent through the true aortic lumen. It is more complex and takes more time. However, there is 90% chance of false lumen obliteration and reduced rates of re-intervention and improved long-term survival [47]. Uchida et al. [47] showed improved survival in the FET group at 5 years (95.3% vs. 69%, $p = 0.03$) and 100% thrombosis of false lumen in FET group compared to 29% patent false lumen in the non-FET group. However, both groups had similar operative mortality. Caution should be exercised that it should be done in high volume centres and by experienced surgeons, as the total duration of hypothermic circulatory arrest can be dramatically increased.

14. Total aortic repair

Deployment of stent in the descending thoracic aorta has its drawbacks. Stent induced false lumen thrombosis activated inflammatory markers like metalloproteinases and proinflammatory cytokines [48], which contribute in the progression of aneurysm by destruction of the extracellular matrix in the aortic wall and neo-angiogenesis. Risk factors for the late development of aneurysm include (i) patent

false lumen, (ii) helicoidal flow distal to the endoprosthesis, (iii) aortic wall shear stress gets modified.

Matalanis [49] has introduced the concept of total aortic repair to prevent the above mentioned complication. Patients presenting with TAAD and a descending thoracic aorta diameter of more than 40 mm can benefited from this approach. The repair involves a “Branch first” total aortic arch repair and surgical ascending aorta repair. Second part included endovascular treatment of descending aorta. It includes covered stent graft deployment in the proximal part of descending aorta and rupture of the intimal flap for the last part of aorta. The rupture is managed with the deployment and dilation under balloon of uncovered stent graft. With this approach, aneurysmal dilatation of the false channel is avoided by the creation of this new aortic channel. But, currently there is no long-term follow-up of this approach.

15. Complications of surgery for ATAD

Based on the NORCAAD registry [50].

1. Bleeding

- i. Major bleeding –39 %
- ii. Reoperation for bleeding 22%.
- iii. Cardiac tamponade –15%

2. Neurological

- i. Stroke 20%.
- ii. Coma 11%.
- iii. Transient ischemic attack 5%.

3. Infections:

- i. Sepsis 10%.
- ii. Deep sternal wound infections 2%.
- iii. Pneumonia 19%

4. Renal

- i. Acute kidney injury 39%.
- ii. Renal replacement therapy 12%

5. Malperfusion

- i. Mesenteric ischemia 6%.
- ii. Myocardial infarction 6%.
- iii. Limb ischemia requiring surgery 4%

6. Reoperations of the aorta

i. Proximal reoperation.

ii. 1 year 0.8%.

iii. 5 years 2.1%

7. Distal reoperations.

i. year 0.8%

ii. 5 years 4.3%

Bleeding is one of the most feared complication from a surgeon's point of view. When blood comes in contact with subendothelial tissue of false lumen, it leads to a coagulopathy. Consumption of coagulation factors and fibrinolysis leads to disseminated intravascular coagulation. Activation and consumption of platelets also contributes to mortality [51]. Patients with pre-operative cardiac mal-perfusion was found to be associated with 30-day mortality of 33% (47). Pre-operative cerebral malperfusion is associated with three-fold increase in risk of stroke. Post-operative stroke and coma occurred in 10–15% and 3–9% patients respectively in one series [46]. Acute kidney injury may occur in 40–55% [50].

16. Long-term follow-up

Long-term post-operative survival in recent years at 5, 10 and 30 years is 84–85, 64–68, and 38%, respectively [52]. Health-related quality of life is lower compared to the general population. There is 32% incidence of depression and post-traumatic stress disorder [53]. Over 50% patients have resistant hypertension on follow-up.

As per the EACTS/ESC 2014 guidelines, a follow-up CT-scan of the aorta is recommended at 1, 6 and 12 months and annually thereafter [54]. There are no recommendations specific to the aortic valve or aortic regurgitation for follow-up. To follow general guidelines, one can perform follow-up echocardiography every 1–2 years for mild regurgitation and annually for moderate and asymptomatic severe regurgitation [55].

17. Conclusion

Type A Aortic dissection is an emergency requiring timely surgical intervention. With improved imaging techniques, an accurate diagnosis can now be made. Open surgical repair techniques have given good long-term results. Endovascular intervention is an emerging less invasive option which can be combined with a surgical approach to give excellent long-term results.

Abbreviations

TAAD	type A aortic dissection
aSCP	antegrade selective cerebral perfusion
RCP	retrograde cerebral perfusion

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